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10/563,195	04/03/2006	Alison Velyian Todd	J&J5215USPCT	6479

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EXAMINER	
BHAT, NARAYAN KAMESHWAR	
ART UNIT	PAPER NUMBER
1634	
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/563,195	TODD ET AL.	
	Examiner	Art Unit	
	NARAYAN K. BHAT	1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *19 December 2008*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-38* is/are pending in the application.

4a) Of the above claim(s) *38* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-37* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *19 December 2008* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *12/19/2008*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☒ Other: *Copy of the Sequencing error report*.

FINAL ACTION

1. This office action is written in reply to applicant's correspondence filed December 19, 2008. Claims 1, 19, 21 and 26 were amended. Applicant's amendments requiring the enzyme differentially modifying alkylated cytosine and cytosine present in the single stranded DNA necessitated the new grounds of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**.

2. Claims 1-38 are pending in this application.

3. Claim 38 is withdrawn from further consideration as being drawn to a nonelected invention and species in the reply filed on May 18, 2007.

4. Claims 1-37 are under prosecution.

Amendments to Claims

5. Amendments to the claims 1, 19, 21 and 26 have been reviewed and entered.

Amendments to Drawing

6. Amendments to the drawings in Fig. 2A-C filed on December 19, 2008 to include SEQ ID numbers as requested by the Examiner in the previous office action has been reviewed and accepted.

Restriction Requirement

7. Applicants traverse the restriction requirement because Opdecamp et al do not teach various elements of the claimed kit (Remarks, pg. 18, paragraph 2). This

argument is not persuasive because as described in the previous office action, Opdecamp et al teaches reagents (EcoR I, HpaII, Buffers) and instructions for using the kit as claimed (Abstract, pg. 172, see Materials and Methods section). Therefore, the technical feature, i.e., a kit, linking group I and II does not constitute a special technical feature as defined by PCT Rule 13.2, because it does not define a contribution over the prior art. Thus, there is no special technical feature linking the group I (claims 1-37) and group II (claim 38), as would be necessary to fulfill the requirements for unity of invention. Therefore, restriction requirement is still deemed proper and is therefore made FINAL.

Specification -Sequence Listing are Not-Compliant

8. Sequence listing provided by the Applicants in the CRF is defective because of errors in the annotation fields. A copy of the error report is enclosed with the action. Please follow reviewer's instructions to correct the errors in the annotation fields.

For the response to this Office Action to be complete, Applicant is required to comply with the requirements for Patent Applications containing nucleotide sequence and/or amino acid sequence Disclosures. Failure to comply with the requirements will be considered as nonresponsive.

Claim Rejections - 35 USC § 112

9. The previous rejections of claims 19, 21 and 26 under 112 Second Paragraph are withdrawn in view of claim amendments.

Claim Rejections - 35 USC § 103

10. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

11. This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

12. Claims 1-4, 14-31, 33 and 35-37 are rejected under 35 U.S.C. 103(a) as being unpatentable over Gitan et al (Genome Research, 2001, 12, 158-164) in view of Bransteitter et al (PNAS, 2003, 100, 4102-4107, cited in the IDS filed 4/3/2006).

Regarding claim 1, Gitan et al teaches a method for detecting the presence or level of alkylated cytosine in a sample of genomic double stranded DNA from an individual, the method comprising following steps.

Regarding step 'a', Gitan et al teaches obtaining a sample of the genomic double stranded DNA from the individual (pg. 162, Methods section, pg. 162, column 2, and paragraph 3).

Regarding step 'b', Gitan et al teaches converting at least one region of the double stranded DNA to single stranded DNA by treating with mild heat and alkali/sodium bisulfite using Intergen kit (Gitan et al, pg. 162, column 2, paragraph 4, Intergen kit, pg. 6, Modification protocol, pg. 7. step II).

Regarding step 'c', Gitan et al teaches that single stranded DNA comprises both methylated (i.e., alkylated) and unmethylated cytosine and bisulfite treatment converts unmethylated cytosine to uracil, whereas methylated cytosine remain unchanged in the single stranded DNA (Fig. 1, pg. 159, column 1, Results section, paragraph 1), which encompasses differentially modifying alkylated cytosine and cytosine present in the single stranded DNA. Methylated cytosine of Gitan et al is the alkylated cytosine as defined in instant claim 35.

Regarding step 'd', Gitan et al teaches determining the level of bisulfite modification of the target region by analyzing converted (i.e., unmethylated) and unconverted (i.e., methylated alleles) alleles on an oligonucleotide array (Fig. 1, bottom panel, pg. 159, results section paragraph 1). Gitan et al are silent about enzyme differentially modifying alkylated cytosine and cytosine present in the single stranded DNA.

Regarding claims 2 and 3, Gitan et al teaches that the single stranded DNA is reacted with the bisulfite under conditions such that the bisulfite reacts substantially only with cytosine (Fig. 1, pg. 159, column 1, Results section, paragraph 1). Gitan et al are silent about enzyme reacting with cytosine present in the single stranded DNA.

Regarding claim 4, Gitan et al teaches that the conversion of the region of the double stranded DNA to the single stranded DNA comprises separating the two strands of the double stranded DNA by treating with mild heat and alkali/sodium bisulfite using Intergen kit (Gitan et al, pg. 162, column 2, paragraph 4, Intergen kit, pg. 6, Modification protocol, pg. 7. step II).

Regarding claim 14, Gitan et al teaches that the determination of the level of bisulfite modification of the single stranded DNA with single stranded comprises analyzing for methylated and unmethylated cytosine sequence variations arising from the enzymatic modification of the target region of the single stranded DNA by the bisulfite (Fig. 4, see the legend).

Regarding claim 15, Gitan et al teach PCR amplification of the selected target region using PCR to obtain an amplified product and analyzing the amplified product by sequence variation (Fig. 1, See the legend for details).

Regarding claim 16, Gitan et al teaches that the analysis of the amplified product comprises subjecting the amplified product to a technique involving the use of probes that bind to specific nucleic acid sequences (Fig. 1, bottom panel).

Regarding claim 17, Gitan et al teaches that the analysis of the amplified product comprises subjecting the amplified product to a polymerase chain reaction technique with nested primers (pg. 162, column 1, and paragraph 1).

Regarding claims 18-21, Gitan et al teaches that bisulfite deaminates cytosine present in the target regions of the single stranded DNA (Fig. 1) but are silent about an enzyme deaminating cytosine.

Regarding claim 22, Gitan et al teaches detecting the presence of methylated cytosine in a ER alpha gene (Fig. 4).

Regarding claim 23, Gitan et al teaches detecting the presence of methylated cytosine in a promoter region, i.e., 5' untranslated region of a gene (pg. 162, column 1, paragraph 1, lines 1-2).

Regarding claim 24, Gitan et al teaches that the level of methylated cytosine comprises hypermethylation (Figs. 4 and 5 and pg. 160, column 1, paragraph 2).

Regarding claim 25, Gitan et al teaches the levels of methylated cytosine in the ER alpha gene is lower in MCF-7 cells than in the MDA-MB-231 cells (Fig. 4, pg. 160, column 1, paragraph 2), which encompasses the levels of methylated cytosine comprise hypomethylation.

Regarding claim 26, Gitan et al teaches that the gene is ER alpha (Abstract).

Regarding claims 27-31, Gitan et al teaches detection of altered level of alkylated cytosine in the target region of the single stranded DNA is a marker for cancer (Abstract and pg. 161, column 1, paragraph 2).

Regarding claim 33, Gitan et al teaches that the variable methylation of the ER alpha CpG island in normal fibroblast strain HFF results in silencing of the ER alpha gene expression (Fig. 5, pg. 160, column 1, last paragraph and pg. 161, column 1, first paragraph), which encompasses the level of methylated cytosine indicating the absence of an altered gene imprinting state.

Regarding claim 35 and 36, Gitan et al teaches that alkylated cytosine is 5-methyl cytosine (pg. 158, column 2, lines 1-2 and pg. 163, column 2, reference of Frommer et al).

Regarding claim 37, Gitan et al teaches double stranded DNA is genomic DNA (pg. 162, Method section, and paragraph 1).

As described above Gitan et al are silent about reacting single stranded DNA with enzyme that differentially modulate alkylated cytosine and cytosine present in the single stranded DNA.

However, an enzyme differentially modifying alkylated cytosine and cytosine was known in the art at the time of the claimed invention was made as taught by Bransteitter et al.

Bransteitter et al teaches a method, wherein an Activation Induced cytidine Deaminase (AID) enzyme that modulates the activity of single strand DNA comprising cytosine and methylated cytosine differentially by 10-fold (Fig. 4b, Abstract and pg. 4106, column 1, paragraph 4).

Regarding claim 18, Bransteitter et al teaches that AID enzyme deaminates cytosine in the target region of the single stranded DNA (Fig. 1, single strand DNA labeled as ssDNA).

Regarding claim 19, Bransteitter et al teaches that Aid enzyme and RNaseA (i.e., combination of enzymes) are employed to differentially modify 5-methyl cytosine (i.e., alkylated cytosine) and cytosine in the target region (Fig. 4B, pg. 4106, column 1, and last paragraph).

Regarding claims 20 and 21, Bransteitter et al teaches that the AID enzyme is a deaminase enzyme having deaminase activity (Fig. 1).

Bransteitter et al also teaches that AID enzyme differentially modifies single stranded DNA containing 5-methyl cytosine than cytosine (Fig. 4b, pg. 4106, column 1, paragraph 4) and further teaches that the deamination is very rapid and completes in minutes (Fig. 4A, pg. 4106, column 1, last paragraph).

Both Gitan et al and Bransteitter et al are interested in understanding the importance of methylation pattern in biological processes and therefore method steps are combinable. As described above, Gitan et al teaches bisulfite treatment for long hours to differentially modulate single stranded DNA comprising unmethylated and methylated cytosine, where as Bransteitter et al teaches an enzyme treatment for minutes to differentially modulate single stranded DNA comprising unmethylated and methylated cytosine. One having the skill in the art would like to use an enzyme for detecting methylation status in a target genome, because it requires less time and does not require additional steps of purifying target as taught by Bransteitter et al.

It would have been prima facie obvious to one having the ordinary skill in the art at the time the invention was made to modify the bisulfite deamination step of Gitan et al with AID enzyme deamination step of Bransteitter et al with a reasonable expectation of success.

An artisan would have been motivated to modify the bisulfite deamination step of Gitan et al with the expected benefit of using AID enzyme for detecting methylation status of the target genome in minutes as taught by Bransteitter et al (Fig. 4A and 4B,

pg. 4106, column 1, paragraph 4), thus expediting the detection of methylation pattern of cancer causing genes using the methylation specific oligonucleotide microarray of Gitan et al.

13. Claims 1 and 4 -13 are rejected under 35 U.S.C. 103(a) as being unpatentable over Gitan et al (Genome Research, 2001, 12, 158-164) in view of Bransteitter et al (PNAS, 2003, 100, 4102-4107) as applied to claims 1 and 4 as above and further in view of Kuhn et al (J. Am. Chem. Soc., 2002, 124, 1097-1103).

Teachings of Gitan et al and Bransteitter et al regarding claims 1 and 4 are described above in section 12.

Regarding claims 5-13, Bransteitter et al teaches generating bubble comprising single stranded region in the double stranded region using partially complementary double stranded DNA (Table 1, legend). Gitan et al and Bransteitter et al are silent about using strand displacing probes to separate the two strands of the double stranded DNA. However, partially separating the two strands of the double stranded DNA with different means including strand displacing probes were known in the art at the time of the claimed invention was made as taught by Kuhn et al.

Regarding claim 4, Kuhn et al teaches a method for opening of the double stranded DNA with PNA openers to partially separate the two strands of the double stranded DNA (Fig. 5A, right panel, pg. 1101, column 1, and paragraph 2).

Regarding claim 5, Kuhn et al teaches a method wherein PNA openers (i.e., strand displacing probes) are used to partially separate the two strands of the double stranded DNA (Fig. 5A, right panel, pg. 1101, column 1, paragraph 2).

Regarding claim 6, Kuhn et al teaches that the strand displacing probes are PNA containing probes (Fig. 5A, right panel, pg. 1101, column 1, and paragraph 2).

Regarding claim 7, Kuhn et al teaches a method wherein the double stranded DNA is opened with PNA openers and further teaches that PNA openers forms triplexes and exposes the displaced DNA strand for binding with other DNA and PNA beacons (Fig. 5A and B, right panel, pg. 1101, column 1, paragraph 2), thus teaching inhibiting annealing of the two strands of the double stranded DNA together once they have been separated to facilitate access to the target region by the enzyme. With regard to the recitation of "to facilitate access to the target region by the enzyme", the phrase is the property of the DNA being at least partially separated, which Kuhn et al teaches.

Regarding claim 8, Kuhn et al teaches hybridizing at least one PNA beacon probe with a strand of the double stranded DNA following separation of the two strands to form a triplex structure, thereby inhibit the annealing of the two strands together (Fig. 5A and B, right panel, pg. 1101, column 1, and paragraph 2).

Regarding claim 9, Kuhn et al teaches that the PNA probe is antisense probe (Fig. 5B, right panel, Fig. 7A, See the legend, pg. 1102, column 2, paragraph 1).

Regarding claim 10, Kuhn et al teaches at least two PNA opener probes are hybridized with the strand of the double stranded DNA, one of the probes hybridizing with a region of the strand downstream of the target region (Fig. 5A, right panel, see the

PNA opener at the right side of the displaced strand) and other probe hybridizing with a region of the strand upstream of the target region (Fig. 5A, right panel, see the PNA opener at the left side of the displaced strand).

Regarding claim 11, Kuhn et al teaches wherein the probe hybridizes with upstream and downstream regions of the strand which flank the target region such that a loop which incorporates the target region is formed in the strand (Fig. 5A, right panel, see the loop formed by two PNA openers).

Regarding claim 12, Kuhn et al teaches that the probe hybridizes with the strand of the double stranded DNA either side of the target region (Fig. 5A, right panel) and further teaches that the probe has a middle region of non-complementary sequence that does not hybridize with the target region such that a loop incorporating the target region is formed in the strand (Fig. 5A, right panel).

Regarding claim 13, Kuhn et al teaches that the middle region of the probe incorporates inverted repeats that hybridize together following hybridization of the probe with the strand of the double stranded DNA (Fig. 5A, right panel).

Kuhn et al also teaches that PNA beacons are insensitive to the presence of salt and DNA-binding/processing proteins and have a great potential as robust tools for recognition of specific sequence within double strand DNA without denaturation and deproteinization of duplex DNA (Abstract).

It would have been prima facie obvious to one having the ordinary skill in the art at the time the invention was made to modify the step of converting double stranded

DNA into single stranded DNA of Gitan et al and Bransteitter et al with the PNA openers of Kuhn et al with a reasonable expectation of success.

An artisan would have been motivated to modify the step of converting double stranded DNA into single stranded DNA of Gitan et al and Bransteitter et al with the expected benefit of using PNA beacons, which are insensitive to the presence of salt and DNA-binding/processing proteins and have a great potential as robust tools for recognition of specific sequence within double strand DNA without denaturation and deproteinization of duplex DNA as taught by Kuhn et al (Abstract).

14. Claims 1 and 32 are rejected under 35 U.S.C. 103(a) as being unpatentable over Gitan et al (Genome Research, 2001, 12, 158-164) in view of Bransteitter et al (PNAS, 2003, 100, 4102-4107) as applied to claim 1 as above and further in view of Opdecamp et al (Nucleic Acids Research, 1992, 20, 171-178).

Teachings of Gitan et al and Bransteitter et al regarding claim 1 are described above in section 12.

Regarding claim 32, Gitan et al teaches the presence of methylated cytosine in human foreskin fibroblast (Fig. 5). Gitan et al and Bransteitter et al are silent about levels of methylated cytosine to indicate the presence or absence of fetal cells. However, levels of methylated cytosine to indicate the presence or absence of fetal cells was known in the art at the time of the claimed invention were made as taught by Opdecamp et al.

Opdecamp et al teaches a method for detecting the presence or level of alkylated cytosine in a sample of genomic double stranded DNA from an individual, the method further comprising the detection of an altered level of alkylated cytosine in fetal liver than in adult liver (Fig. 5, Compare the pattern of fetal and adult liver), which encompasses the level of the alkylated cytosine to indicate the presence of fetal DNA.

Opdecamp et al also teaches that higher gene expression in fetal than in adult cells is due to methylation pattern of unmethylated site in the non-coding portion of the gene in fetal and adult cells (Abstract).

It would have been prima facie obvious to one having the ordinary skill in the art at the time the invention was made to use the method of detecting methylation pattern in cancer cells of Gitan et al and Bransteitter et al and apply said method to detect the presence of fetal cells as suggested by Opdecamp et al with a reasonable expectation of success.

An artisan would have been motivated to detect fetal cells by the method of Gitan et al and Bransteitter et al with the expected benefit of detecting genes that are expressed in fetal cells are also expressed in cancer cells as taught by Opdecamp et al (Abstract).

15. Claims 1 and 34 are rejected under 35 U.S.C. 103(a) as being unpatentable over Gitan et al (Genome Research, 2001, 12, 158-164) in view of Bransteitter et al (PNAS, 2003, 100, 4102-4107) as applied to claim 1 as above and further in view of in view of Paulson et al (J. Virol., 1999, 73, 9959-9968).

Claim 34 is dependent from claim 1. Teachings of Gitan et al and Bransteitter et al regarding claim 1 are described above in section 12.

Regarding claim 34, Gitan et al teaches detecting the presence of methylated cytosine changes in multiple CpG island loci in breast cancer (Abstract). Gitan et al and Bransteitter et al are silent about the presence of alkylated cytosine to indicate the presence or absence of a pathogen or microorganism.

However, the presence of alkylated cytosine to indicate the presence or absence of a pathogen or microorganism was known in the art at the time of the claimed invention was made as taught by Paulson et al, who teaches that the EBV is the etiologic agent of infectious mononucleosis, i.e., a pathogen (pg. 9959, column 1, paragraph 1) and further teaches that the presence of EBV is characterized by the presence of its methylated promoter sites (Fig. 3, pg. 9964, column 2 paragraph 2). Paulson et al also teaches that EBV usurps the host cell directed methylation system to regulate pathogen gene expression and thereby establish a chronic infection (Abstract).

It would have been prima facie obvious to one having the ordinary skill in the art at the time the invention was made to use the method of detecting methylation pattern in cancer cells of Gitan et al and Bransteitter et al and apply said method to detect the presence of pathogen as suggested by Paulson et al with a reasonable expectation of success.

An artisan would have been motivated to detect pathogen by the method of Gitan et al and Bransteitter et al with the expected benefit of detecting expression of genes

that are induced by pathogen EBV (Paulson et al, Abstract, pg. 9959, column 1, and paragraph 1) are also induced or repressed in cancer cells.

Response to remarks from the Applicants

Rejections under 35 U.S.C. § 102(b)

16. Applicant's arguments with respect to claims 1-3, 14, 19, 22-25, 27-33, 35 and 37 as being anticipated by Opdecamp et al have been fully considered but are moot in view of withdrawn rejection and new grounds of rejection set forth in this office action necessitated by claim amendments (Remarks, pgs. 20-21).

Rejections under 35 U.S.C. § 103(a)

17. Applicant's arguments with respect to claims 1 and 4-13 as being unpatentable over Opdecamp et al in view of Kuhn et al have been fully considered but are moot in view of withdrawn rejection and new grounds of rejection set forth in this office action necessitated by claim amendments (Remarks, pg. 21, last paragraph).

Applicant's arguments with respect to claims 1 and 14-17, 22, 23 and 26 as being unpatentable over Opdecamp et al in view of Gitan et al have been fully considered but are moot in view of withdrawn rejection and new grounds of rejection set forth in this office action necessitated by claim amendments (Remarks, pg. 22 and pg. 23, first paragraph).

Applicant's arguments with respect to claims 1 and 34 as being unpatentable over Opdecamp et al in view of Paulson et al have been fully considered but are moot in view of withdrawn rejection and new grounds of rejection set forth in this office action necessitated by claim amendments (Remarks, pg. 23 paragraphs 2-4).

Applicant's arguments with respect to claims 1, 18, 20-21 and 35-36 as being unpatentable over Chaudhuri et al in view of Bransteitter et al have been fully considered but are moot in view of withdrawn rejection and new grounds of rejection set forth in this office action necessitated by claim amendments (Remarks, pg. 23 last paragraph, pg 24).

Conclusion

18. No claims are allowed

19. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL.** See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of

the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Narayan K. Bhat whose telephone number is (571)-272-5540. The examiner can normally be reached on 8.30 am to 5 pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram R. Shukla can be reached on (571)-272-0735. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Narayan K. Bhat/

Examiner, Art Unit 1634

/Ram R. Shukla/

Supervisory Patent Examiner, Art Unit 1634